THE RELATIONSHIP BETWEEN HOP BIOMECHANICS, FUNCTIONAL PERFORMANCE, AND TALAR CARTILAGE DEFORMATION IN HEALTHY INDIVIDUALS

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ABSTRACT

Devon Nicole Stroup: The Relationship Between Hop Biomechanics, Functional Performance, and Talar Cartilage Deformation in Healthy Individuals
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Lateral ankle sprains are a very common injury in sport, however often go underreported and mismanaged. This leads to a cascade of events including chronic ankle instability (CAI) development. People with CAI often exhibit altered biomechanics, as well as degenerative joint changes when compared to healthy individuals that can lead to post-traumatic osteoarthritis (PTOA). It is unknown if biomechanics are linked to cartilage deformation, which stands as a sensitive marker for joint health and PTOA development. Research is also limited in ultrasound sensitivity to talar cartilage deformation after loading. Before identifying this relationship and establishing ultrasound as a sensitive imaging technique of deformation in pathologic individuals, we must first investigate healthy individuals. Therefore, our overall purpose is to determine how talar cartilage deforms in healthy individuals after a standardized single leg hopping loading protocol, and to explore how hop biomechanics and functional performance influence deformation after this protocol.

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LIST OF ABBREVIATIONS

ASIS	Anterior Superior Iliac Spine
ATFL	Anterior Talofibular Ligament
CAI	Chronic Ankle Instability
CFL	Calcaneofibular Ligament
IC	Initial Contact
LAS	Lateral Ankle Sprain
MRI	Magnetic Resonance Imaging
OA	Osteoarthritis
PTOA	Post Traumatic Osteoarthritis
US	Ultrasound
VGRF	Vertical Ground Reaction Force

CHAPTER I: INTRODUCTION

Lateral ankle sprains are one of the most common injuries sustained during athletic activities.[1] It is estimated that more than 28,000 ankle sprains occur every day in the United States.[2] Although lateral ankle sprains have a high prevalence, the perception of the injury is often seen as inconsequential.[3] As a result, this injury goes significantly underreported and it is estimated that as many as 55% of people who sustain an acute lateral ankle sprain do not seek medical care.[4] The benign perception of this injury perpetuates the negative sequela of events that occur due to the injury being poorly managed.

Poorly managed lateral ankle sprains can result in a high recurrence of injury. It is estimated that up to 74% of initial acute ankle sprains result in subsequent injury, residual symptoms, and feelings of instability.[5] Mechanical instability and functional instability due to recurrent ankle sprains has been termed as chronic ankle instability (CAI) and can lead to many negative consequences, including feelings of "giving way," difficulty and apprehension on uneven surfaces, and the possible presence of pain.[6] Perhaps one of the most significant consequences of CAI is the early degenerative changes in the joint due to unbalanced loading.[7] It is estimated that up to 78% of patients with long term CAI develop osteoarthritis (OA) beyond 10 years[7], with post-traumatic osteoarthritis (PTOA) representing 70% of all OA in the ankle.[8]

Degeneration of articular cartilage due to direct or indirect injury, or PTOA, is a significant health concern and results in significant disability. As many as 4 out of 5 cases of ankle OA are

the result of previous injury to the joint, with the target population being at least 10 years younger than those who develop primary ankle OA.[9] Saltzman et al. found that the reported degree of physical impairment in patients with ankle OA is equal to the impairment associated with severe medical problems including congestive heart failure and end-stage kidney disease.[10] In addition, ankle OA results in a large financial burden, accumulating around $12 billion in associated costs annually in the United States.[11] With its high prevalence, high cause of disability, and high cost, lateral ankle sprains and the resultant sequelae (i.e. CAI and PTOA) present a major health concern.

Another consequence that coincides with the negative sequela of ankle injury is altered joint kinematics. Patients with CAI and early degeneration of the ankle joint have been shown to possess altered kinematics that increase the ground reaction forces exerted through the ankle.[12] As stated previously, early degenerative changes can be seen in the ankle joint due to altered loading.[7] Bischof et al. observed that patients with ankle instability possessed an increased peak cartilage strain and an anteromedial shift in position of maximum strain of the talar articular cartilage.[13] Increased cartilage contact strain has been theorized to contribute to cartilage damage.[14] Although no direct links between altered kinematics and osteoarthritis have been found, it is plausible to say that one might influence the other due to both being present in patients with CAI, ankle cartilage degeneration, and PTOA.

One of the longstanding measures of joint health is cartilage behavior. Many studies have looked at cartilage thickness, cartilage content, and cartilage deformation in the knee and ankle. MRI methods are the gold standard for cartilage imaging, however they are very expensive and difficult to reproduce with high fidelity. Ultrasound is a cost-effective alternative that has

identified cartilage deformation and thickness measures in the knee, and due to its success in the knee it may be a good, cost-effective alternative tool to look at ankle joint cartilage health [15,16,17].

Before we can understand the effects of altered kinematics on cartilage deformation in pathologic individuals, we must first define normal cartilage deformation in a healthy population and understand how biomechanical alterations influence cartilage deformation. Therefore, the overall purpose of this study is to explore how hop biomechanics, in healthy individuals, influence talar cartilage deformation after a standardized single leg hopping loading protocol. To achieve the overall purpose, we will answer the following research questions:

1. Does talar cartilage deform following a standardized hop protocol in healthy individuals?

   Hypothesis: We hypothesize that the thickness measures of talar cartilage using ultrasonography will significantly change immediately after a standardized hop protocol.

2. How do lower extremity biomechanical outcomes correlate to cartilage deformation outcomes in healthy individuals?

   Hypothesis: We hypothesize that sagittal plane joint angles and vertical ground reaction forces during hopping will correlate with cartilage deformation.

3. How do functional tasks correlate to talar cartilage deformation outcomes in healthy individuals?

   Hypothesis: We hypothesize that functional task performance will correlate with cartilage deformation magnitude.

CHAPTER II: REVIEW OF THE LITERATURE

Epidemiology

Lateral Ankle Sprain

Ankle injuries can be very serious and represent a prevalent health concern. Estimates suggest that 28,000 ankle injuries occur every day in the US alone[2], accounting for 20% of all injuries treated in emergency facilities.[1] Ankle sprains alone, which happen to be the most common type of ankle injury, make up 10% of all emergency room visits in the United States.[18] However, it is approximated that 55% of individuals who sustain an ankle sprain do not seek medical care, and therefore injuries may be significantly underreported.[4] In athletics, ankle sprains are the highest reported injury, accounting for up to 45% of all athletic injuries.[1] Lateral ankle sprains in particular make up 85% of all ankle sprains.[1] Risk factors predisposing someone to lateral ankle sprains are divided into intrinsic and extrinsic categories. Intrinsic factors include age, sex, height, body mass index (BMI), history of previous injury, fitness level, limb dominance and girth, flexibility, muscle strength, proprioception, reaction time, postural stability, anatomical alignment, foot morphology, and inadequate rehabilitation.[19,20] Extrinsic factors include specific sport and position, level of competition, shoe type, playing surface, and the use of external restraints.[21] However, history of previous ankle sprain presents as the best predictor for recurrent ankle sprain.[22]

Due to the high prevalence of lateral ankle sprains, this injury poses a large financial burden on afflicted individuals. A study by Shah et al.[23] reported that on average, an emergency room visit for a lateral ankle sprain costs about $1,211, equating to greater than

$1.1 billion in total yearly health care costs. However, this $1.1 billion only represents the acute cost of emergency room visits, not acute management by family physicians or other health care providers. On top of the cost, acute ankle sprains have been shown to lead to many other consequences for the affected individual.

Chronic Ankle Instability

Research has shown that up to 74% of individuals with a previous history of ankle sprain suffer from a negative sequela of residual symptoms, recurrent ankle sprains, and/or feelings of instability long after the initial injury[5], also termed chronic ankle instability (CAI).[24] Just as acute injury poses a significant financial burden, chronic joint injury is responsible for extensive medical costs as well. It is estimated that up to 87% of all costs resulting from lateral ankle sprains comes from future rehabilitation and therapy, time lost from work, and treatment for long-term symptoms, all issues associated with CAI.[23] If not treated appropriately, chronic ankle instability may result in chronic pain, chronic and pathological adaptations, osteochondral lesions of the talus, and premature osteoarthritis.[25]

Post-traumatic Osteoarthritis

Post-traumatic osteoarthritis (PTOA) stands as one of the biggest contributors to long term disability.[26] Osteoarthritis (OA) is the most frequent cause of rheumatic complaints[27], and PTOA represents 70% of all foot and ankle osteoarthritis.[8] PTOA is a result of an injury to the joint[27], such as an ankle sprain[28], as a result of overuse, overload, or damage of other joint tissues.[29] The disease then manifests, slowly degenerating the articular cartilage over time. Symptoms of ankle PTOA include pain in the talocrural joint line, limited range of motion, and decreased function, ability to work, and ability participate in leisure activities due to pain.[30] PTOA has been shown to develop in up to 78% of people with chronic ankle instability beyond

10 years.[31] Unlike joint degeneration in the knee and hip, PTOA in the ankle tends to occur in younger patients[30], and results in faster functional loss, with end stage disease progression reaching its height within 10 to 20 years after the start of the lesion.[32] According to Bischof et al., damage to the anterior talofibular ligament and calcaneofibular ligament during an acute ankle sprain may be associated with the development of PTOA.[13] In a study conducted by Valderrabano et al., researchers found that in a group of 247 consecutive patients treated for ankle arthritis, lateral ligament damage was the primary contributor to the development of ligamentous PTOA.[14] PTOA in the ankle generates insurmountable associated costs due to the disability it creates. In the United States alone, PTOA is responsible for over $3 billion in direct health care expenses[11,28], and over 12 billion in total related costs.[11] Overall, the negative sequela from acute ankle sprains and chronic ankle instability to ankle joint degeneration and PTOA are shown to be very common, very expensive, and very difficult to treat, leading to significant long-term disability.

To fully understand what needs to be done to help treat and prevent this progressively degenerative condition, we must first understand the contributing factors and the pathophysiology of ankle PTOA to understand what needs to be addressed and eventually mitigate the long-term disability caused by this disease.

Biomechanical Alterations

Altered kinematics have been shown to be present in people affected by ankle joint pathology and degeneration, and have long been associated with the etiology of OA.[26] During the normal gait cycle, people with CAI have been shown to have a lower walking velocity, lower cadence, shorter step length, and wider base of support.[33] Individuals suffering from CAI also

exhibit increased inversion, lateral shift of body weight, increased hip flexion during terminal swing to mid stance, reduced hip extension, increased knee flexion during terminal stance to initial swing, and slower weight transfer at the beginning and end of the stance.[34,35] Individuals with perceived instability produce compensatory altered gait kinematics in order to complete dynamic tasks, and these alterations reflect deficits caused by their instability.[33,34] These compensatory mechanisms during gait contribute to higher vertical ground reaction forces and loading rates. In a study conducted by Bigouette et al., authors found that among individuals with CAI, impact peak forces, average loading rates, and active peak forces were all higher when compared to healthy individuals during running because of altered kinematics.[12] As seen in a study conducted by Bischof et al., individuals with CAI exhibited greater cartilage contact strains when compared to healthy ankles, and these areas of greater strain corresponded to the region of talar cartilage where OA was clinically observed.[13] However, no correlations between peak vertical ground reaction forces and increased cartilage contact strains have been established.

Deficits have also been shown during dynamic landing tasks, which is a functional movement performed in many sports.[36] During landing, individuals with CAI exhibit a lower angle of eversion and abduction in the ankle joint than healthy individuals.[37] It has been shown that increased eversion and dorsiflexion in the ankle may be associated with shock absorption strategies that reduce vertical ground reaction forces.[37] Individuals with CAI alter their mechanics to offset instability, however this compensatory movement in turn reduces shock absorption. In a study done by Lee et al., athletes with CAI were shown to have a reduced shock absorption capacity adjusted ankle joint angle compared to controls during single-leg

drop landing when fatigue was induced in both groups.[37] In this study, CAI participants also displayed lower eccentric work of the knee.[37] This study showed that CAI individuals have higher dependence on their ankle than controls in post-fatigue drop landing, which may increase their risk of recurrent ankle injury and perpetuate the negative sequela of ankle joint injury and degeneration.[37] In addition, these authors suggest that participants with CAI have a decreased ability to control impact forces from single-leg drop landing due to altered landing strategies after induced fatigue, which may increase their risks of potential injury in the ankle joint.[37] Other studies have shown that altered biomechanics from injury put an individual at risk for progressive joint degeneration.[21,38] Goreham-Voss et al. found that abnormal loading associated with injury and instability resulted in increased peak stresses and loading rates on the articular cartilage when looking at cadaveric ankles.[39] Cartilage loading rates over 25 MPa/s have been shown to cause cell death, and in this study all ankles exceeded this loading rate except for the fully in-tact model, representing a healthy ankle.[39] Altered loading has also been thought to contribute to the development of chondral lesions and arthritis and be detrimental to cartilage health.[14,21] Bischof et al. found that individuals with CAI possess an increased peak cartilage strain and an anteromedial shift in position of maximum strain in the talus, perpetuating cartilage damage sustained during the initial injury.[13] Ultimately, altered contact strains due to altered kinematics could contribute to cartilage degeneration and the development of ankle OA.[13] Although no direct link has been observed between altered kinematics and altered cartilage activity, both have been seen in individuals with CAI and ankle joint degeneration, so it is plausible to say there is a correlation. Because of the observed

differences in cartilage activity when comparing healthy cartilage to pathologic cartilage, cartilage has been used to measure the status of an individual's joint health.

Measures of Joint Health

Cartilage Thickness

In a normal, healthy ankle, ankle cartilage thickness ranges from less than 1 to 2mm and has a contact area of 350mm^2 in load bearing areas.[40] Ankle cartilage is much thinner than that of the knee and hip, and also has a smaller cartilage contact area, which results in higher forces per area when the lower extremity is loaded.[40] However, due to the compositional differences in ankle cartilage compared to knee cartilage, the tissue responds differently to loading and has been shown to resist degradation at higher rates than knee articular cartilage, possibly accounting for the lower prevalence of ankle OA.[40] Several studies have looked at cartilage contact strains in healthy individuals using MRI. MRI has been labeled as the gold standard for assessing knee cartilage thickness[41], and has been used in studies to look at ankle cartilage thickness.[42] MRI allows for the visualization of intraarticular pathologies that are not evident on plain radiography[43], and is the best tool for looking at deep chondral and subchondral lesions.[44]

Cartilage Content

Understanding cartilage composition is also critical to understanding potential mechanisms and behaviors that can represent early biomarkers for OA. Articular cartilage consists of chondrocytes and an extracellular matrix composed of collagen, water, and proteoglycans.[45] The combination of water (fluid phase) and the dense collagen and proteoglycans (solid phase) of the extracellular matrix allows articular cartilage to efficiently distribute load through the joint and gives the articular cartilage its low friction properties.[45]

The fluid phase provides the matrix with the ability to dissipate load and the hydraulic pressure within the fluid phase provides very important load support and protects the solid phase from significant stresses.[45] The collagen and proteoglycans resist compression, and in normal articular cartilage proteoglycans are arranged in such a way to maintain a porous-permeable solid matrix.[45] However, OA has been directly linked to the loss of proteoglycan content.[45] In OA cartilage, there is a breakdown of the proteoglycan structure, leading to a more penetrable solid matrix.[45] This increased permeability leads to a decrease in the hydraulic pressure and causes a decrease in compressive stiffness, thereby increasing the stress from loading.[45]

Two types of compositional MRI are T2 and T1ρ mapping, which have been used to assess articular cartilage properties. T2 and T1ρ mapping techniques are relatively new methods for the compositional examination of articular cartilage.[46] T2 mapping identifies the water content and collagen fiber status of articular cartilage.[43,44] An increase in T2 relaxation values can be seen as a biomarker for early cartilage damage and destruction of the collagen fiber network.[47] T1ρ relaxation time is sensitive to changes in proteoglycan content in the articular cartilage and increased relaxation times represent less proteoglycan content.[43] In the knee, studies have shown that increased T2 and T1ρ relaxation times are positively correlated with the severity of OA.[48,49]

In the ankle, disparities in T2 values have been shown to exist in pathologic individuals when compared to healthy participants. A study conducted by Golditz et. al found that individuals with classified functional ankle instability showed uneven loading in the ankle and a significant increase in T2 values when compared to controls.[47] Another study conducted by Lee et al. found that individuals who have partial or full tears of their anterior talofibular ligament

(ATFL) had increased T2 values at the medial anterior, lateral anterior, and lateral center compartments of their talar cartilage when compared to individuals with an intact ATFL.[50] As ligamentous injury may stand as a predisposing factor to the development of PTOA in the ankle[13], this study lends insight into the correlation between ATFL injury and cartilage degeneration.[50]

As for T1ρ methods, research has shown that individuals with CAI have higher T1ρ relaxation times compared to healthy individuals.[51] Research has also shown that greater T1ρ relaxation times, meaning less proteoglycan density, positively correlate with greater talar cartilage thickness attributed to increased water content (swelling) from cartilage breakdown in individuals with CAI.[52] However, most MRI methods, including T2 and T1ρ mapping, are expensive, not available to all patients at all times, and not easily available for consecutive measurements of cartilage status.[16]

Cartilage Deformation

Understanding cartilage deformation is crucial to developing an understanding of cartilage function and biomechanical factors that may relate to cartilage degeneration.[53] Many studies have identified relative cartilage thicknesses in response to loading, and most studies have focused on examining cartilage thickness in the knee. Measures of cartilage thickness using MRI provides an estimate of overall cartilage structure, however it is not the most sensitive marker for OA development.[54] In the earliest stages of OA, there is a change in cartilage composition without significant declines in cartilage thickness.[54] Therefore, identifying the response of cartilage structure, or cartilage deformation, to acute loading has been thought to provide a more accurate substitute measure of cartilage composition, because

cartilage composition determines how cartilage deforms.[55,56] Most research involving the examination of cartilage deformation and recovery rate after activity has been conducted at the knee. In the healthy knee, research has shown that cartilage deformation occurs at similar magnitudes during walking and running conditions.[15] In contrast, during a small frequency, high amplitude drop landing task which comprised of 100 vertical drops in 30 minutes, cartilage deformation was significantly lower when compared to a running task.[56]

In a study by Li et al.[53], researchers examined healthy human ankle joint articular cartilage deformation using MRI under different constant loads while standing. They found that the cartilage deformation rate reached a peak value at the first second after loading, and continued to increase until about 50 seconds of constant loading. However, after about one minute of loading, the deformation rate dramatically decreased and the cartilage deformation and contact area remained at a relatively constant high. The authors explained that at initial contact, the contact area was small so the rate of peak cartilage deformation was high. However, as the joint continued to increase contact area, there was a resultant decrease in deformation rate.[53]

In a study conducted by Van Ginckel et al., researchers examined ankle cartilage deformation using MRI during an in vivo exercise task of 30 closed kinetic chain knee bends in one minute reaching maximal dorsiflexion in healthy individuals.[26] After the in vivo exercise task, authors found significant deformation in talar cartilage, and the cartilage recovered within 30 minutes post-exercise.[26] However, no current studies have examined talar cartilage deformation or recovery rate during a dynamic hopping task.

As stated previously, MRI methods are expensive and not easily accessible.[16] Ultrasound (US) has become a recent method of great interest in quantifying pathologic cartilage in the knee due to ease and low cost, accessibility and good reproducibility.[15,16,17] In a study conducted by Schmitz et al. that compared the use of US to MRI imaging techniques, ultrasound cartilage thickness measures in the knee were significantly positively correlated with MRI thicknesses.[16] In another study conducted by Harkey et al. that looked at ultrasound as a tool for identifying medial femoral cartilage deformation, authors found that US imaging is sensitive to medial femoral cartilage deformation immediately following walking and running in healthy individuals.[15] Overall, US appears to be beneficial to the researcher and clinician due to lower cost, greater availability, and access compared to MRI techniques.[16] Although US has not been extensively used to look at talar cartilage deformation, due to its success in the knee it may be a sensitive, cost-effective alternative tool to look at ankle joint cartilage health.

In conclusion, the sequela of events leading to PTOA is detrimental. Being able to identify predisposing factors and early biomarkers of the disease, including altered biomechanics and cartilage behavior, will allow us to eventually find early intervention methods to decrease the negative impact PTOA has on individuals. Many studies have looked at static weight bearing, walking and running to evaluate cartilage deformation and recovery rate. However, the literature is lacking in highly dynamic tasks, such as jump landing and hopping. Single limb landing is a common functional task in sport, and a very common mechanism for ankle injury.[36] When people return to their sport, jumping and single/double leg landing is often the mechanism for recurrent ankle sprain leading to development of CAI[36], which is known to be a predisposing factor for ankle degeneration and PTOA[25]. Before we understand what cartilage deformation in

pathologic patients looks like during a functional landing/hopping task, we must first understand how functional landing/hopping affects healthy cartilage deformation. Therefore, the purpose of this study is to identify the biomechanics and deformational behavior of talar articular cartilage in healthy individuals after a dynamic single limb hopping task. These results will lend themselves as a basis for future studies looking at altered kinematics and cartilage deformation in the pathologic population.

CHAPTER III: METHODS

3.1 Research Design

This descriptive laboratory study seeks to identify if talar cartilage deforms after a single leg hopping task, how hop and jump biomechanics correlate with talar cartilage deformation, and how performance on functional tasks correlate with talar cartilage deformation. The independent variable for research question 1 is time. There are no independent variables for research questions 2 or 3 as it is a correlation study. Talar cartilage deformation is a dependent variable for all questions. Hop and jump biomechanics are dependent variables for question 2, and performance on the functional tasks is a dependent variable for question 3.

3.2 Participants

Participants in the study consisted of thirty healthy, physically active individuals and we recruited them in person or via email. Inclusion criteria for this group consisted of being between 18-35 years of age, no previous history of lower extremity ligamentous injury, no chronic ankle instability or balance disorders, no history of lower extremity surgery, no history of concussion or head injury in the past six months, no history of cardiac condition or stroke, not currently pregnant, and individuals who are recreationally active (i.e. participate in at least 3, 30 minute sessions of physical activity per week). Exclusion criteria consist of failing to meet the inclusion criteria.

3.3 Instrumentation

The instruments used in this study included the VICON Motion Capture System (VICON, Oxford Metrics, Oxford, UK), Bertec force plates, and a Phillips Lumify portable ultrasonography system on a Microsoft tablet. The motion capture system included 10 high-speed VICON cameras synchronized with three force plates[57] positioned within the center of the capture volume. Kinematic data were collected at a sampling frequency of 120 Hz. Kinetic data were collected at a sampling frequency of 1200 Hz and low-pass filtered at 75 Hz. We placed retroflective markers placed on the acromion processes, sternal notch, sacrum, anterior superior iliac spines (ASIS), greater trochanters, mid-thighs, lateral and medial femoral condyles, tibial crests, lateral and medial malleoli, calcanei, and first and fifth metatarsals of each participant. We used ultrasonography to capture the cross-sectional area of the talar cartilage. A Phillips Lumify portable ultrasonography system captured images at a frequency of 25 Hz, gain of 50, and depth of 3.0 cm.

3.4 Procedures

This study was conducted over the course of two, 75-minute sessions. As part of a larger study, participants completed a survey to determine eligibility as control subjects. After determining eligibility, participants read and signed the university approved informed consent form. We then recorded the the participants' age, height, and weight. We also asked participants, "if you were to kick a soccer ball, which foot would you use?" Participants then performed all tasks on their chosen limb. During the first session, participants performed various assessments as part of a larger study.

Second, we completed a biomechanical analysis of the subject. Participants changed into spandex shirts and shorts and completed a static standing trial by standing with feet shoulder width apart and arms across his or her chest after being marked with the aforementioned anatomical markers. Using Visual3D software, we created a kinematic model of the pelvis, thigh, shank, and foot from the static trial.[57] Then, the subject completed a series of single leg hopping and jump landing for biomechanical analysis. For each assessment, participants completed a series of 3 practice trials to familiarize themselves with the task. For the single leg hop, the participant was positioned 24 inches away from the force plate. They jumped from his or her chosen limb onto the center of the right force plate, landing on the same limb, and stabilizing for roughly two seconds. They completed this task a total of 5 times. Next, participants completed 5 jump landings from a twelve-inch box onto the force plates. Participants completed both tasks with simultaneous biomechanical analysis using the VICON System. Participants then had the markers removed.

Additionally, participants completed four functional tests including the side hop test, the figure-of-8 test, the single leg hop test, and the crossover hop test on their chosen limb. For the side hop test, we instructed participants to hop on the chosen limb laterally over a 30-cm distance. Hopping laterally 30 cm and back to the starting location constituted 1 repetition, and each participant completed 10 repetitions as fast as possible. A repetition was only successful if the participant's foot landed outside of the 30-cm distance marked by tape. For the figure-of-8 hop, participants completed single leg hops in a figure-8 pattern, twice around a 5-meter course marked by cones as fast as they could. We recorded both tests to the nearest 10^{th} of a second. For the crossover hop test, the patient performed 3 hops as far as possible crossing

over a 15cm wide strip marking on each hop and maintain landing after the 3rd hop for 2 seconds. The participant had to maintain stabilization at the end of the trial to be considered a successful trial the first of the 3 hops is lateral with respect to the direction of the crossover. The participants completed 3 successful trials and the best of the 3 was recorded. Lastly, the single limb hop for distance required the participant to stand on one limb and hop as far forward as possible, landing on the same limb along a 15-meter strip. To be a successful trial, the participant had to maintain stabilization for 2 seconds. The participant completed 3 successful trials and the best of the 3 trials were recorded. We recorded both tests to the nearest 10th of a centimeter[58]. We randomized the order of task completion for each participant to minimize the influence of fatigue on performance.

Participants then returned for a second session for cartilage deformation analysis. Participants sat in a long-sit position for one hour to unload the cartilage of the ankle. [15] We measured knee and ankle joint angles at 140 degrees for optimal image capture. As part of a larger study, we took images of the ankle and knee using the Lumify ultrasound system at 15-minute increments. At the end of the one hour unloading period, we captured another set of ultrasound images of the knee and ankle by placing the probe longitudinal to the joint line and rotating to best show the articular cartilage image.[15,27,51]

Participants then placed their shoes back on, were seated in a chair, and rolled in a chair to align themselves with the starting position of the hop loading protocol. The loading protocol consisted of a total of 60 single-leg hops on their chosen limb. We instructed participants to stabilize each hop for about 2 seconds. Participants hopped 12 times in one direction across the testing space, and then turned around and hopped back 12 times. Participants completed

this 5 times for a total of 60 hops, with each hop being about 28 inches in distance. If participants lost balance, we allowed the participant to place his or her other foot down to help regain their balance before returning to a single limb stance as quickly as possible. At the completion of the 60th hop, participants were seated in the chair again, and rolled back to the table to have another series of talar cartilage images taken in a manner identical to the pre-test.

We analyzed sagittal hip, knee, and ankle kinematics and vertical ground reaction force kinetics. We extracted these variables using the VICON Nexus system and Visual3D Software. We cropped all trials, confirmed and labeled the presence of all markers, and created body segments in VICON. We then imported trials into Visual3D for biomechanical and kinetic analysis. If needed, we used a fill technique before running the data through a custom written MATLAB code.

We measured and analyzed cartilage images using ImageJ Software. Variables extracted included lateral, medial, and overall area and length. We imported each image into ImageJ, set the measuring scale to pixels/cm, and adjusted each picture to a 0 degree angle for uniform analysis. First, we measured the overall length of the talar cartilage from medial peak to lateral peak of the cartilage. We then marked the halfway, lateral edge and medial edge points using the multi-point tool. We defined the lateral/medial regions as 50% between the middle of the image and the lateral/medial "peaks." We then measured lateral and medial areas by tracing the outline of each segment of the cartilage with the polygon function on ImageJ. We defined the lateral/medial area as the area from the middle point to the lateral/medial edge. We took care to trace the outline of the cartilage area only included cartilage (appearing as black on the

image). If needed, we adjusted outlines for precision. We calculated the overall area as the sum of the lateral and medial areas. Finally, we measured the length of the lateral segment by drawing a line to measure from one end of the traced area to the other end. We repeated the process for the medial length, and measured an overall length once again. We measured and analyzed a total of 3 pre-loading images and 3 post-loading images for each participant to get average values. From these average values, we calculated lateral, medial and overall normalized cross-sectional areas by dividing the respective pre- and post- area by width for each segment for the statistical analysis.

3.5 Statistical Analyses

To achieve RQ1, we conducted three dependent T-tests to compare pre- to post-deformation for lateral, medial, and normalized cross-sectional areas. Because values were not normally distributed for RQ1, we conducted a non-parametric Wilcoxon Signed Ranks Test to determine if cartilage deformed after a standardized hop loading protocol. In addition, we conducted pre-to post Cohen's d effect sizes and 95% confidence intervals. To achieve RQ2, we conducted Pearson Product Moment Correlations among the specific peak vertical ground reaction force (vGRF), biomechanical, and cartilage deformation measures. To achieve RQ3, we conducted Spearman Correlations among the functional test scores and cartilage deformation measures as well, due to values not being normally distributed. We set statistical significance to alpha=0.05.

CHAPTER IV: RESULTS

4.1 Demographics

Thirty healthy, recreationally active participants met the inclusion and exclusion criteria for this study. There were twenty-three females and seven males included in the study. Twenty-eight participants chose the right as his or her dominant limb, and two chose the left. The age, height and weight of participants were 20.52±2.37 years, 170.39±8.63 cm, and 65.98±13.07 kg, respectively.

4.2 Dependent T-Tests

For aim one, five out of the six values for cartilage thickness showed significance: pre-lateral $p<.05$, post-lateral $p<.01$, pre-medial $p=.20$, post-medial $p<.05$, pre-overall $p<.05$, post-overall $p<.05$. These values indicate that the data are not normally distributed. Therefore, we conducted a non-parametric Wilcoxon Signed Ranks Test to determine if cartilage deformed after a standardized hop loading protocol. We found a statistically significant difference between pre- and post-measures for lateral ($p<.001$), medial ($p<.001$), and overall ($p<.001$) normalized cross-sectional areas. Means, standard deviations, and effect sizes can be found in Table 4.1. Average change scores and percent changes pre- to post-loading can be found in Table 4.2.

Table 4.1: Means, Standard Deviations, and Effect Sizes

Mean ± SD (mm)	Pre	Post	P-values	Cohen's d Effect Size
Lateral	0.4646 ± 0.1428	0.4283 ± 0.1369	<0.001	0.7497
Medial	0.5034 ± 0.1436	0.4654 ± 0.1306	<0.001	0.8402
Overall	0.4873 ± 0.1383	0.4496 ± 0.1297	<0.001	0.9825

Table 4.2: Average Change Scores and Percent Changes Pre- to Post-Loading

	Average Change mm (decrease)	Percent Change % (decrease)
Lateral	0.0376 ± 0.0494	7.48%
Medial	0.0410 ± 0.0472	7.30%
Overall	0.0398 ± .0402	7.65%

4.3 Correlations

For aim two, we ran the Shapiro-Wilk Normality Test and the significance values indicated that the variables were normally distributed, so we chose to run Pearson product-moment correlations for these variables. All correlations and p-values between cartilage deformation and single leg hopping and jump landing biomechanics can be found in tables 4.3 and 4.4.

For single leg hopping biomechanics, there was a significant correlation between loading rate and change in lateral cartilage thickness ($r= -.412$, $p=.024$). As loading rate decreased, cartilage deformation increased. There were also significant correlations between sagittal ankle angle at initial contact and changes in lateral ($r=-.525$, $p=.003$), medial ($r= -.550$, $p=.002$), and overall ($r= -.644$, $p<.001$) thicknesses. As sagittal joint angle decreased, or as plantarflexion increased, at initial contact, cartilage deformation increased. There were no statistically

significant correlations found between normalized peak vertical ground reaction force (vGRF), sagittal knee and hip joint angles at initial contact, or peak joint angles and changes in cartilage thickness.

For jump landing, there were significant correlations between loading rate and changes in lateral ($r=-.576$, $p=.001$), medial ($r=-.379$, $p=.039$), and overall ($r=-.598$, $p<.001$) thicknesses. As loading rate decreased, cartilage deformation increased. There were no statistically significant correlations found between normalized peak vGRF, sagittal joint angles at initial contact, or peak joint angles and changes in cartilage thickness.

Table 4.3: Correlation Coefficients and P-values for Single Leg Hopping Biomechanics vs. Cartilage Deformation

	Lateral Change	Medial Change	Overall Change
Peak vGRF	r= -.321(p=.084)	r: .114(p=.549)	r: -.120(p=.529)
Loading Rate	**r: -.412(p=.024)**	r: -.174(p=.358)	**r: -.361(p=.050)**
Sagittal Ankle Angle (IC)	**r: -.525(p=.003)**	**r: -.550(p=.002)**	**r: -.644(p<.001)**
Sagittal Knee Angle (IC)	r: -.055(p=.774)	r: -.023(p=.906)	r: -.040(p=.832)
Sagittal Hip Angle (IC)	r: .027(p=.887)	r: .176(p=.353)	r: .093(p=.625)
Peak Ankle Dorsiflexion	r: .109(p=.568)	r: -.041(p=.831)	r: .033(p=.862)
Peak Knee Flexion	r: -.132(p=.487)	r: .117(p=.537)	r: .004(p=.965)
Peak Hip Flexion	r: -.188(p=.320)	r: .080(p=.675)	r: -.099(p=.603)

Table 4.4: Correlation Coefficients and P-values for LESS Jump Landing Biomechanics vs. Cartilage Deformation

	Lateral Change	Medial Change	Overall Change
Peak vGRF	r: -.234(p=.213)	r: -.195(p=.303)	r: -.242(p=.197)
Loading Rate	**r: -.576(p=.001)**	**r: -.379(p=.039)**	**r: -.598(p=<.001)**
Sagittal Ankle Angle (IC)	r: -.238(p=.206)	r: -.078(p=.680)	r: -.203(p=.282)
Sagittal Knee Angle (IC)	r: -.182(p=.336)	r: -.153(p=.419)	r: -.202(p=.284)
Sagittal Hip Angle (IC)	r: .230(p=.221)	r: .321(p=.084)	r: .308(p=.098)
Peak Ankle Dorsiflexion	r: -.157(p=.408)	r: -.103(p=.589)	r: -.168(p=.375)
Peak Knee Flexion	r: -.295(p=.114)	r: -.282(p=.131)	r: -.342(p=.065)
Peak Hip Flexion	r: -.048(p=.799)	r: -.028(p=.885)	r: -.053(p=.781)

For aim three, normality tests showed significance values that indicated data were not normally distributed. Therefore, we conducted Spearman's rank correlations to determine a relationship between functional hop test performance and changes in cartilage thickness after a standardized hop loading protocol. Table 4.5 shows Spearman correlation values and significance levels. There was a statistically significant correlation between single leg hop distance and changes in lateral (ρ=.442, p=.015), and overall (ρ=.400, p=.029) thicknesses. As hop distance increased, cartilage deformation increased. There was a statistically significant correlation between crossover hop distance and changes in lateral (ρ=.508, p=.004), and overall

(ρ=.442, p=.014) thicknesses. As hop distance increased, cartilage deformation increased. Lastly, there was a statistically significant correlation between side hop time and changes in lateral (ρ=-.453, p=.012), and overall (ρ=-.385, p=.036) thicknesses. A decrease in side hop test time is correlated with an increase in cartilage deformation. There was no statistically significant correlation between the figure 8 hop test and lateral, medial or overall normalized cross-sectional area deformation. There was no statistically significant correlation between any of the functional performance tests and the medial normalized cross-sectional area deformation.

Table 4.5 Correlation Coefficients and P-Values for Functional Performance vs. Cartilage Deformation

	Lateral Change	Medial Change	Overall Change
Figure 8	ρ=-.356 (p=.054)	ρ= -.154 (p= .417)	ρ=-.293 (p= .116)
Single Leg Hop	**ρ= .442(p=.015)**	ρ= .239 (p= .203)	**ρ= .400(p= .029)**
Crossover Hop	**ρ= .508(p= .004)**	ρ= .269 (p= .150)	**ρ= .442(p= .014)**
Side Hop	**ρ= -.453(p= .012)**	ρ= -.220 (p= .244)	**ρ- -.385(p= .036)**

CHAPTER V: DISCUSSION

The purpose of this study was multi-faceted. The first aim of this study was to determine whether or not talar cartilage deformed after a standardized hop loading protocol in healthy individuals. The second and third aims were to determine if there was a relationship between 1) lower extremity biomechanics and talar cartilage deformation, and 2) functional performance and talar cartilage deformation. We hypothesized that 1) talar cartilage would deform after a standardized hop loading protocol, 2) lower extremity biomechanics would correlate with talar cartilage deformation, and 3) functional task performance would correlate with talar cartilage deformation. We found statistically significant differences between pre- and post-thickness measures in lateral, medial and overall areas of the measured talar cartilage images. This indicates that healthy talar cartilage does in fact deform following a hop loading protocol and supports our a priori hypothesis. We also found significant correlations between plantar flexion angle at initial contact during a single leg hopping task and talar cartilage deformation, and between loading rate and lateral cartilage deformation. Additionally, we found significant correlations between loading rate during a jump landing task and talar cartilage deformation. These findings support our a priori hypothesis. Lastly, we found statistically significant correlations between three of the four functional performance tasks and cartilage deformation magnitude which supports our a priori hypothesis for Aim 3.

Deformation Post-Loading

Mechanical loading is believed to play a pivotal role in maintaining joint health.[59] In the knee, healthy articular cartilage deforms in response to mechanical loading, and due to the low permeability of the articular cartilage matrix, time is required for the cartilage to restore itself to an unloaded state.[60] This is why measurements of relative cartilage thickness after loading have been used as a measure of joint health. In the earliest stages of OA, there is a change in cartilage composition without significant declines in cartilage thickness.[54] Therefore, identifying the response of cartilage structure, or cartilage deformation, to acute loading has been thought to provide a more accurate surrogate measure of cartilage composition, because cartilage composition determines how cartilage deforms.[55,56] Thus, we would anticipate that talar cartilage would deform following mechanical loading.

Research has shown substantial cartilage deformation in healthy individuals after different in vivo impact conditions using MRI analysis techniques.[26,61] In a study conducted by Van Ginckel et. al. researchers investigated percent change of talar cartilage deformation after performing 30 bilateral knee bends in 12 healthy individuals using MRI.[26] They analyzed the 3D images pre-loading, as well as at 4 time points post-loading and determined a 10.41% average volume decrease immediately after loading. In a follow up study, the authors[61] used 13 healthy individuals to complete 4 different in vivo tasks and again quantified cartilage deformation using 3D MRI. Tasks included 30 bilateral knee bends, 30 unilateral knee bends, 2-minute unilateral stance, and 10 single leg drop jumps which corresponded to an 8.3%, 7.7%, 14.6%, and 12.5% decline in cartilage volume respectively. Their research suggests that static loading, or sustained loading over time, produces greater deformation magnitude than dynamic loading.

To date, there is limited research to demonstrate that US is capable of quantifying cartilage deformation following a loading protocol in general [15,62,63,64] and no studies demonstrating this ability at the ankle. However, research has demonstrated the validity of US based measures of cartilage morphology. Schmitz et al. noted that ultrasound cartilage thickness measures in the knee were significantly positively correlated with MRI thickness measures.[16] Further, research has demonstrated femoral cartilage deformation using US based thickness measures following a variety of dynamic loading protocols.[15,62,63] Consistent with the existing literature, we showed a 7.65% mean decrease in overall normalized cross-sectional area following a dynamic hop protocol using US based measures. Thus, our results demonstrate that US can capture comparable talar cartilage deformation in healthy individuals as a more sensitive MRI analysis. This suggests that US may be a suitable tool to assess cartilage behavior in a more clinical setting.

Research to date consistently demonstrates cartilage deformation following loading but the magnitude of difference varies slightly (~2-5%). Differences could be attributed to types of loading, duration of loading, and the use of ultrasound vs. MRI. However, the relatively small differences between studies further supports the claim that ultrasound imaging could be a clinical surrogate of MRI in measuring talar cartilage health metrics. Future research should focus on determining reliability and reproducibility of ultrasound imaging specifically in the ankle, as well as if US is sensitive to differences in deformation between healthy and pathologic groups.

Biomechanical Correlates

Our study examined relationships between various biomechanical outcomes and cartilage deformation magnitude. Previous research has suggested that healthy individuals may exhibit greater plantarflexion at initial contact because this allows for contact forces to be distributed through a greater range of motion and allow for reduction of force.[65] Caufield and Garrett also found that healthy individuals exhibit greater plantarflexion during landing.[66] Similarly, static (i.e. sustained) loading elicits greater cartilage deformation[59], as it is associated with lower loading rates. Lower loading rates, because of greater plantar flexion positioning at IC, appears to result in greater deformation of the talar cartilage.

Cumulatively, these results make intuitive sense. Healthy individuals are able to modulate impact forces (i.e. lower loading rates via greater plantar flexion) which increase cartilage strain. While this may seem like a negative result, it is important to remember that cartilage deformation is also a force absorption technique in the body and greater deformation is an appropriate physiologic response in this situation. This complex interaction is likely the reason why positive correlations among lower loading rates, greater plantar flexion at IC, and greater cartilage deformation magnitude were observed in our healthy sample.

While associations were noted with IC plantar flexion during a hop task, no such association was noted for IC plantar flexion during the jump landing task. This may be due to the nature of the loading protocol used. The single leg hop task mimicked our loading protocol, whereas the double leg jump landing did not. This may be particularly important for future research and clinical stress tests as it appears that only biomechanical profiles representative of the loading protocol influence cartilage deformation. Future research is needed to test this

hypothesis and determine how gait biomechanics influence cartilage deformation following walking and how jump landing biomechanics influence cartilage deformation following a jump landing loading protocol.

Interestingly, no significant correlations were found between IC sagittal knee or hip joint angles in either task and cartilage deformation magnitude. Research has shown that healthy individuals have more variability in hip and knee flexion during single leg jumping prior to IC and through stance phase.[67] Thus the lack of associations may be due to a more variable kinematic pattern of the proximal joints within a healthy population. Similarly, no significant correlations between peak joint measures or vertical ground reaction forces and cartilage deformation magnitude were noted. Because we saw correlations between loading rates but not peak joint angles, this may support the notion that loading rate is more important to cartilage behavior than overall load or an overall peak joint angle. However, this remains largely speculative due to lack of studies looking specifically at peak joint angles during dynamic tasks, which makes it difficult to draw conclusions about whether or not there may be a relationship to cartilage deformation magnitude. If rate of movement is in fact more important than overall movement in the joint when examining the way cartilage deforms, this could also explain the lack of associations among peak vGRF and cartilage deformation magnitude in our sample of healthy individuals. Future research is needed to confirm our initial results and better determine the roles of peak force and joint angle versus loading and movement rates.

Functional Performance

Our study found that as performance improved in certain functional tasks, there was greater cartilage deformation magnitude. In table 5.1, we have compared our means to

previously reported means on the tasks in question to demonstrate the generalizability of our results within a young adult healthy population. With the exception of the figure 8 hop test, our mean functional performance scores are very similar to those found in previous studies observing healthy individuals, which supports that our sample is representative of a healthy population.

Table 5.1: Comparing Means of Task Performance Scores Across Literature

	Single Leg Hop (m)	Crossover Hop (m)	Figure 8 Hop (s)	Side Hop (s)
Current Study	1.3 ±0.3	3.5 ± 0.9	12.7 ± 2.1	10.1 ± 2.9
Caffrey et al[69]		3.2 ± 0.3	11.0 ± 0.5	9.9 ± 0.4
Sharma et al[70]	1.51 ± 0.1		7.17 ± .02	9.08 ± 0.52
Docherty et al[58]	1.48 ± 0.32		6.98 ± 1.01	9.09 ± 0.91

Stated previously, we found that better functional performance and loading rates correlate with greater deformation magnitude in the lateral and overall aspects of the cartilage in our healthy sample. These correlations may mean that these functional tasks could serve as sensitive cartilage stress tests. From a clinical perspective, this could mean that clinicians could establish baseline functional movement performances of patients and potentially identify individuals who are at risk of altered cartilage loading, which has been linked to early degenerative changes in the joint.[7] Functional performance is a more bedside tool than biomechanical outcomes. Eventually, these stress tests may allow clinicians to initiate preventative rehabilitation programs to improve functional performance and mitigate the potential for altered cartilage loading.

Our study found that better functional performance and *lower* loading rates correlate with greater cartilage deformation magnitude. Intuitively, one may think that better performance, i.e. jumping farther and faster, is likely to lead to higher impact forces and higher loading rates, which has been observed in the CAI population during landing. This contradicts our findings. If true, then better functional performance should have resulted in less cartilage deformation. Thus, we hypothesize that healthy individuals who perform better may have performance strategies that allow them to better attenuate force and lower loading rates. Better performance in healthy individuals may be due to better, more adaptive neuromuscular and sensorimotor landing strategies that could also be advantageous for cartilage health. The assessment of these neuromuscular strategies was beyond the scope of this investigation but should be an area of future research.

Limitations

There were several limitations in this study. For example, the subjects recruited were self-reported as healthy and recreationally active, which could result in self reporting bias. Our sample was also heavily female and right-limb dominant, which may not be representative of the larger population. Additionally, the assessor was not blinded to whether or not the cartilage images were pre or post loading. We also only looked at the ankle cartilage immediately after loading, so we did not capture cartilage recovery rate, which is also a measure of joint health. Lastly, we only took images of talar cartilage at one joint angle. This allowed us to only see one 2D cross-sectional area of the subjects' cartilage which may not be representative of deformation and health across the entire talar dome.

Clinical Implications and Future Research

This study observed that healthy cartilage does deform in response to a standardized hop loading protocol when measured using ultrasound imaging. The findings of this study also indicate that cartilage deformation magnitude correlates with increased plantarflexion at initial contact and lower loading rates, as well as better performance on certain sport-specific functional tasks. Because we have seen positive correlations between 1) biomechanics , 2) functional performance scores, and 3) cartilage behavior found in healthy individuals, our results support a relationship between the way individuals load their joint and how their talar cartilage responds to that load. Although more research needs to be done, this study may act as a foundation for studies investigating talar cartilage deformation in pathologic individuals, as well as the relationship between biomechanics, functional performance, and cartilage deformation magnitude in pathologic individuals. For example, previous research has shown differences in loading rates between how healthy individuals and those with CAI during a running task.[12] A recent systematic review also found that healthy individuals exhibited lower loading rates than CAI individuals during unilateral jump landing tasks.[71] Additionally, those with CAI have been shown to have altered biomechanical profiles[33,34,35] and functional performance scores relative to uninjured controls.[58,67,68] When these group differences are coupled with our observed associations, it is likely that those with CAI have differences in cartilage deformation behavior. While this investigation provides further support for the use of ultrasound imaging as a clinical surrogate to MRI measures of cartilage health, determining if US is sensitive to differences in cartilage deformation in pathological groups relative to uninjured controls is the next step.

Conclusions

This study aimed to bridge the gap in the literature between lower extremity biomechanics, functional performance, and healthy cartilage deformation of the talus. Before developing a full understanding of the effects of altered kinematics on cartilage deformation in pathologic individuals, we needed to establish normal cartilage deformation measures in a healthy population and understand how biomechanical correlates influence cartilage deformation. Based on our results, talar cartilage deforms, as measured via US, following a hop loading protocol and this deformation occurs across the entire width of the talar dome. Further, cartilage deformation magnitude is associated with key biomechanical parameters during a hop but not a jump landing task. Additionally, cartilage deformation magnitude in the lateral and overall aspects of the talus is also associated with better performance on select functional tasks. In total, the results provide meaningful support for US to be used as a clinical surrogate for MR based measures of ankle joint cartilage health and may provide insight into healthy biomechanical and cartilage deformational profiles, laying the foundation for future research investigating pathologic individuals.

APPENDIX: DATA COLLECTION SHEET

Number:_____ Date:_____

DOB:_____ Height:_____ Weight:_____ Sex: _____

Dominant limb: _____ Involved limb: _____

Data collection time: Session1: Session2:

Functional testing

	Figure 8	Single leg hop	Cross-over hop	Side hop
1				
2				
3				

Biomechanics
Hopping

	Hopping
1	
2	
3	
4	
5	

Jump landing (50% of height)

	LESS
1	
2	
3	
4	
5	

US measures

Positioning

Degrees (140)	Distance
Ankle	

Hopping protocol

60 Single leg hops (Control1H001)

	0			15			30			45			60			Post		
Ankle	1	2	3	7	8	9	13	14	15	19	20	21	25	26	27	31	32	33
Knee	4	5	6	10	11	12	16	17	18	22	23	24	28	29	30	34	35	36

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